Information

New Mexico's First Case of Plague

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THE FIRST CASE of plague in a human in the United States was reported from San Francisco in 1899.[1] From 1899 through 1926 California accounted for 80 percent (394/494) of human plague cases in the continental United States and the remainder occurred in other Pacific or Gulf Coast states (Florida, Louisiana, Texas, Washington).[2] Since 1927, however, plague cases have been more frequently reported from inland compared with coastal states. Thus, from 1949 through 1979, New Mexico (85 cases), Arizona (22), Colorado (13), Utah (3), Idaho (1), Nevada (1) and Wyoming (1) have together accounted for 85 percent (126/149) of the human plague cases in the American West.[2]

When a disease first appears in a geographic area, it may be difficult to recognize, especially if the disease is uncommon. The following narrative explains how plague, a disease new to the Southwest, was first diagnosed in New Mexico in 1949.

Report of a Case

DR POND: Near Cerro, in Taos County, a 9-year-old boy and his brother were returning from an unsuccessful rabbit hunt when the brother killed a prairie dog by hitting him in the head with a rock. Only a sick prairie dog could have been caught in this fashion. The other brother picked up the animal and they proceeded homeward. Nothing happened to the older brother, but the next day the 9-year-old boy became very ill and was brought to our office in Taos. He was seen by one of our associates and given penicillin for a presumed infected blister on his left hand, with lymphadenitis in the axillary nodes. He became much worse during the night and was taken to the Holy Cross Hospital (Taos) emergency room. He was promptly admitted to the hospital with a temperature over 40.6°C (105°F). I saw him the next morning. He had an angry red sore on his left hand and large, partially fluctuant nodes in the left axilla. There was little if any evidence of lymphangitis.

The boy appeared very sick and the thought of plague entered my mind. I had seen a case of plague in San Francisco in 1936. That patient was a veterinary surgeon, exposed constantly to animals and their fleas. He recovered in spite of a two- or three-day delay in establishing the diagnosis. As I questioned various experts about plague, I learned that the infestation of San Francisco had started with shipboard rats getting ashore. The wild rodents had become infected in turn. The experts also told me that the infestation in wild rodents would spread eastward and eventually involve New Mexico where I intended to practice. Consequently, when I moved to New Mexico later in 1936, I expected eventually to see a case of plague. In fact, before 1949 I had sent specimens on a couple of less suspicious cases to the state laboratory in Albuquerque for plague testing and always received negative reports. The laboratory accused me of crying wolf.

I aspirated the 9-year-old boy's bubo and a direct smear showed an organism that could well be *Yersinia pestis*. The rest of the material was cultured and injected into guinea pigs. The following day I was called to the biological laboratory, to be greeted by several dead guinea pigs alongside microscopes with smears of the deadly plague bacillus. Up to this point I had still thought of plague as a farfetched diagnosis. I suddenly realized that it was here and now. Not fully appreciating the role of fleas, I began to worry about my own exposure to this deadly bug. Perhaps a little *snakebite medicine* relieved some of the worry.

The state laboratory met the plane with a sample from the patient's axillary bubo. They kindly reported a suspicious smear by phone and the next day had a positive diagnosis with their guinea pigs. In the meantime I wired San Francisco for an update on antibiotic treatment of plague. The answer was to use a combination of streptomycin and sulfadiazine. A regimen of these was started immediately; the boy began to improve almost at once and in a couple of days he was well on the way to an uneventful recovery. This was the first case of plague in New Mexico.

Comment

Plague infection in animals in this country appears to have started at the end of the 19th century and the beginning of the 20th century when rat plague appeared in port cities on the Pacific Coast.[1-4] Then, plague apparently invaded wild rodent populations close to port areas (first documented in California in 1908).[1] Once established in sylvatic rodents, plague spread rapidly to rodent populations throughout large areas of the West,[1-4] as documented by US Public Health Service surveys conducted in the 1930s and 1940s.[1] The plague expert's prediction that wild rodent plague would reach areas like New Mexico was confirmed in 1938 when it was found in rock squirrels *(Spermophilus varie-*

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gatus) and prairie dogs *(Cynomys gunnisoni)* in Catron County.[1]

Report of this first New Mexico plague case, with the attendant publicity, probably facilitated recognition of two subsequent but unrelated plague cases in New Mexico in 1949. From 1949 through 1982, a total of 114 cases with 22 fatalities were reported from New Mexico. Recognizing a rare disease is always a difficult accomplishment; consideration of plague in New Mexico and in other western states remains a challenge.[5,6]

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Newer Forms of Insulin

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THE PURITY of insulin has changed dramatically in the past ten years and indications for the use of the newer and more purified forms are being established. There are now 42 insulin products in the United States, and at least six more products for human use will probably be approved soon by the Food and Drug Administration (FDA).

The term "purified insulin" is somewhat misleading because as each new purification step was adopted for use in commercial preparation of insulins, the product was described as "purified." Current FDA terminology, however, allows only insulins containing less than 10 ppm of proinsulin and other related residues to be called purified insulin. These insulins became available in the United States in 1980 and were an improvement over the conventional insulins, which contained up to 30,000 ppm of impurities.

The purified insulins are made from either beef or pork insulin, as are the standard insulins, though many of the standard insulins are combinations of both. Immunogenically, pork is preferable as an insulin source because only 1 of the 51 amino acids making up the insulin molecule is different from that in the human insulin molecule, compared with three different amino acids of the beef insulin molecule. The species differ enough immunologically to cause greater antibody formation in those persons exposed to purified beef insulin than in those treated with purified pork insulin.

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In the United States insulins now range in purity from 50 ppm to less than 10 ppm, with the purified insulins available only in concentrations of U 500 (500 units per ml) (for insulin-resistant states) and U 100. Other insulins can be obtained in U 40 concentrations, and insulin diluents are available for mixing concentrations that may be desired for use in insulin pumps or for making small doses such as for infants.

The indications for use of purified insulins are as follows:

- *Lipoatrophy* (pitting at the insulin injection site). This complication disappears when a patient is switched to purified pork insulin. Administration of purified insulin must be continued after resolution of the lipoatrophy, however, because reexposure to the less pure insulin, even though at a different injection site, can cause a reappearance of the atrophy that is resistant to subsequent treatment with the more purified forms. An immune component of lipoatrophy is suggested by the high coincidence of local allergy and lipoatrophy (15 percent) and the reduction of the atrophy with local injection of steroids.[1]
- *Insulin hypertrophy* (fat hypertrophy at injection site). This complication may also have an immune component. It does not respond as predictably to therapy with purified insulins, however, with improvement occurring in only about half of the patients.[2]
- *Allergic reactions to insulin.* Allergic reactions take several forms. Local allergic responses to insulin are varied and can occur within 15 minutes to 2 hours after the injection or they can be delayed and not appear for 4 hours or more after the injection. Common allergies often occur one to four weeks after institution of insulin therapy or within a few days of reinstitution of insulin and are characterized by a hard, red induration at the injection site. The longer acting insulins are more likely to induce this response. Protamine, the agent that slows the action of isophane insulin suspension (NPH insulin), is itself a significant antigen. Systemic allergic responses can also occur and are characterized by urticaria or angioedema, or both, which is mediated by IgE antibodies. More than 60 percent of persons having systemic allergic reactions to insulin have had intermittent insulin therapy and 37 percent are allergic to penicillin. Systemic allergic reactions to insulin are potentially life threatening. If continuing insulin therapy is necessary, a patient should be desensitized in hospital using purified insulins according to the protocol accompanying the desensitization kit, and then maintenance doses of insulin must be administered to maintain the desensitization.
- *Antibody-mediated resistance to insulin.* Such resistance occurs equally in both sexes and usually appears within five years of institution of therapy (in 66 percent of these, insulin resistance develops within the first year). Onset is usually gradual and 75 percent of insulin-resistant patients are older than 40 years of age. About half of these patients need more than 1,000 units of insulin a day and occasionally more than 25,000 units a day is needed during a resistant phase.